NASA TT F-9572

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N67 10197

FACILITY FORM 602

(ACCESSION NUMBER) 29 (PAGES) | (THRU) 1 (CODE) | (NASA CR OR TMX OR AD NUMBER) | 04 (CATEGORY)

Journal of the Physiological Society of Japan,
Vol.23, No.3, pp.133-146, 1961.

GPO PRICE $ _____

CFSTI PRICE(S) $ _____

Hard copy (HC) 2.00

Microfiche (MF) .50

ff 653 July 65

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
WASHINGTON OCTOBER 1965

STUDY OF THE FUNDAMENTAL VIBRATION OF THE ARTERIAL PULSE WAVE **/133

Jiro Sato*

The cause of the fundamental vibration of the arterial pulse wave was studied on the arterial pressure pulse of aorta and femoral artery in dogs and rabbits. The correlation between the following factors was investigated.

1) $\lambda \sim C$ 2) $\lambda \sim \alpha S$ 3) $\lambda \sim W$ 4) $\lambda \sim \frac{W}{C}$ 5) $W \sim \frac{P_2}{P_1}$ 6) $\frac{P_1 - Pd}{P_0 - Pd} \sim \frac{P_2}{P_1}$

(The meaning of the notation is explained in Fig.1.) It is observed that the wavelength of this fundamental vibration changes with the pattern of ejection and the dynamical state of the vascular system. It is concluded that this vibration mainly originates from the ejection of blood from left ventricle to arterial system.

In general, in man as in the monkey, dog, cat, rabbit, and other animals, the so-called central arterial wave is unremarkable in regions near the main part of the aorta, although a distinct arterial vibration wave form is noted in the great peripheral arteries, such as the femoral and radial arteries. This is what is termed the fundamental vibration, and there has long been discussion of many kinds, proceeding from dynamic viewpoints, about the causes of this vibration. The most convincing explanation is that it is an intravascular vibra-

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** Numbers in the margin indicate pagination in the original foreign text.

tion due to the sudden surge of blood that accompanies the closing of the semilunar valves, and on this basis much study has been devoted to the relationship between the wavelength of this vibration and the length of the bloodvessel in which it occurs (Bibl.3, 6, 8, 9, 10, 11, 14, 21, 22, 23).

Since this type of fundamental vibration is considered to be a vibration in an elastic tube, it is hardly necessary to say that its period and damping conditions are subject to the influence of the various dynamic properties of the circulatory system, such as the elasticity of the vascular walls, the diameter of the lumen, the state of branching, the viscosity of the blood, and the like. As Hatakeyama (Bibl.11) has argued, the dynamic constitution of the circulatory system is unusually complex, so that the simple calculation of the wavelength and similar quantities, regarding the vibration as taking place in an open or closed tube, without further refinements, involves the danger of serious errors.

This makes it necessary, first of all, to assume some relationship between the various indices of the properties of the circulatory system and the various elements of the fundamental vibration, based on a simple dynamic model, and then to inquire whether or not this relationship agrees with the phenomena actually observed. If the explanation of the second peak accompanying closure of the semilunar valves [Wiggers (Bibl.24)] is to be considered tenable, then there must be a close relationship between the time of the second peak of the vibration and that of the closure of these valves. Moreover, if, in addition to dynamic conditions of this kind, the biological vascular reactions and similar factors also make a substantial contribution [Wehn (Bibl.20)], then the various above-mentioned dynamic conditions must of necessity be totally unable to give a complete explanation. There have as yet, however, not been many re-

ported studies of the relationships between the fundamental vibration and the various dynamic factors from this point of view. I have therefore made reliable measurement of the dynamic quantities in animal experiments and have thus elucidated the essential nature of the fundamental vibration.

I. EXPERIMENTAL METHOD

The experimental animals used were dogs and rabbits. The dogs were anesthetized with about 0.3 mg/kg of pentobarbital sodium, and the rabbits with about 1 gm/kg of urethane. The blood pressure of the central region of the aorta was measured through the left carotid artery, considered representative of the blood pressure in the central arteries. The peripheral arterial blood pressure was measured in the dogs through the A. profundus femoris, while in the rabbits it was measured directly, after section of the femoral artery in the lower part of the thigh. Although this interruption of the arterial blood flow, for instance in the carotid artery, and, in the rabbits, in the femoral artery, resulted in certain deviations of the measured arterial blood pres- /134 sure from that under normal circulatory conditions, no questions related to the object of this work arose so long as the constant dynamic conditions were maintained. The pressure was measured by an electrical method through a high-stability high-compliance capacitance manometer with a low-compliance cannula system.

The animals were treated as follows:

1) Administration of adrenaline

A 1:10,000 solution was intravenously injected at the rate of about 1 cc in 10 sec. The rabbits received 1 cc and the dogs 4 cc. Immediately after completion of the injection, and thereafter at 10 sec intervals for 1 min,

seven measurements were made and recorded.

2) Administration of acetylcholine

The same procedure as in 1) was followed.

3) Section of the vagus nerve

Two measurements were made 1 min and 1 min 30 sec after section, separately for right and left.

4) Stimulation of central end of vagus nerve

Using an electron tube stimulator with a square wave of 2 msec pulse width, stimuli of the following frequency and intensity were applied for 20 sec:

	Frequency	Intensity
Rabbits:	10 cps	20 v
	100 cps	20 v
	100 cps	30 v
Dogs:	10 cps	20 v
	50 cps	20 v
	200 cps	20 v

Measurements were made and recorded at 0, 10, and 20 sec after the beginning of the stimulation and 10 sec after its end.

5) Stimulation of peripheral end of vagus nerve

Stimulation was applied under the following conditions using the same wave form as above.

	Frequency	Intensity
Rabbits:	10 cps	5 v
	20 cps	5 v
	10 cps	20 v
Dogs:	30 cps	5 v
	30 cps	6 v
	30 cps	10 v

Measurements were made and recorded 0, 5, 7, 10, and 20 sec after the beginning of stimulation.

6) Section of n. depressor cordis

Measurements were made and recorded 1 min after bilateral section.

7) Stimulation of n. depressor cordis

Stimulation was applied under the following conditions, using the same wave form as above.

Frequency	Intensity
10 cps	10 v
10 cps	20 v
10 cps	30 v

Measurements were made and recorded 5, 7, 10, 15, and 20 sec after the beginning of stimulation.

8) Tension on lower limb

The left thigh was pulled by a cord for 30 sec, and measurements were made and recorded 15 and 30 sec after beginning of the tension.

9) Asphyxia

After insertion of a tracheal cannula into the trachea, its orifice was closed for 20 or 30 sec, and measurements were made and recorded 15 and 30 sec after the beginning of such closure.

10) Bloodletting

The time required for the blood pressure to fall to zero, after withdrawing 5, 10, 15, 20, or 30 cc of blood, was recorded.

The blood pressure of the same animal, untreated, was also measured several times. It was not a primary object of the present study to investigate how the dynamic conditions vary after such treatments. Our object was rather to investigate how the condition of the circulatory system changes and to study the dynamic laws that should be established. In view of this object, therefore, from the recorded blood-pressure curves of the two arteries in question we first of

all measured the following quantities:

A. Aortal Blood-Pressure Curve

1. Systolic pressure ..... P_s

2. Diastolic pressure ..... P_d

3. Systolic period ..... S

4. Diastolic period ..... D

5. Period of arterial wave ..... T

B. Femoral Artery Blood-Pressure Curve

6. Systolic pressure ..... P'_s

7. Diastolic pressure ..... P'_d

8. First amplitude of fundamental vibration ..... P_1

9. Second amplitude of fundamental vibration ..... P_2

[With reference to Frank (Bibl.8, 9), as shown by Fig.1b, the amplitude of /135
the blood pressure is measured from the crest of the fundamental vibration to its trough; P_1 is the height of the first peak, and P_2 that of the second.]

10. Period of fundamental vibration .. T'

11. Time during which peak 1 of the fundamental vibration persists ..... S'

C. Other Directly Measured Elements

12. Arterial pulse-wave propagation time ..... Δt

(The time lag between the aortic pressure wave and the femoral artery pressure wave was measured at pulse heights 1/5 of their amplitudes.)

13. Cross sectional area of primitive aorta ..... Q

14. Anatomic length of aorta L

15. Aortic transmission distance (pulse conduction length)..... ℓ

(If L_2 is the distance from the site of insertion of the cannula in the carotid artery to the point at which this artery branches from the aorta, and L_1 is the distance from this branching point of the carotid artery to the point of insertion of the cannula in the femoral artery, then $\ell = L_1 - L_2$.)

D. Elements Used in Calculations

16. Arterial pulse wave velocity..... $C = \ell/\Delta t$

17. Fundamental wavelength..... $\lambda = cT'$

18. Mean blood pressure..... P_m

(Although the exact mean blood pressure would have to be expressed by $P_m = \frac{\phi P dt}{T}$, since the mean blood pressure P_m is expressed as a function $P(t)$ of the time t, we have here taken approximately $P_m = \frac{P_s + P_d}{2}$.)

19. Cardiac output..... V_s

$$V_s = \frac{0.5\ QTS\ (P_s - P_d)}{D\rho C}$$

(According to the Broemser-Ranke formula)

$$\rho = 1.07.$$

20. Mean ejection velocity..... i

$$i = \frac{V_s}{T}$$

21. Effective peripheral resistance... W

$$W = \frac{P_m}{i}$$

Also, as required, we calculated $\frac{\lambda}{L}$, cS, $\frac{W}{c}$, $\frac{P_s - P_d}{P_s' - P_d'}$, $\frac{P_2}{P_1}$, and $\frac{S'}{S}$.

Investigation of the correlation between these measured and calculated

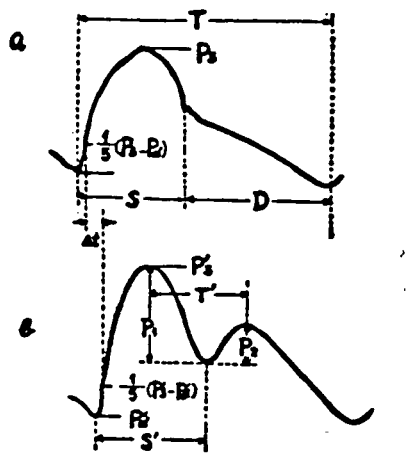

Fig.1a, b Schematic Representation of Various Factors of Aortic a) and Femoral b) Pulse Wave

quantities from the viewpoint of the present work proceeded by studying the correlation coefficients between the quantities:

$$1)\ \lambda \sim C;\quad 2)\ \lambda \sim cS;\quad 3)\ \lambda \sim W;\quad 4)\ \lambda \sim \frac{W}{c};\quad 5)\ W \sim \frac{P_2}{P_1};$$

$$6)\ \frac{P_s - P_d}{P'_s - P'_d} \sim \frac{P_2}{P_1}$$

II. EXPERIMENTAL RESULTS

Table 1, for illustrative purposes, gives one each of these measured and calculated values for the dog and the rabbit, always expressed in CGS units. It is hardly necessary to mention that P_s, P_d, the arterial pressure, and the circulation are all increased by the administration of adrenaline. The value of c is also increased, but T' is decreased, and consequently $\lambda = cT'$ is not too greatly increased and may even show a tendency to decrease. The administration of acetylcholine, in contrast, decreases P_s, P_d and the arterial pressure, while T' increases, so that λ does not decrease very much and may even, in many cases,

increase.

Section of the vagus nerve increases P_s and P_d and decreases c. The value of T' has a tendency to increase, but no very marked effect on λ was noted. Stimulation of the central end of the vagus nerve, depending on the stimulation conditions used, may either raise or lower the blood pressure, and the changes in c, T' and λ may also be either up or down. It is obvious that stimulation of the peripheral end of the vagus nerve induces marked brachycardia and a fall in blood pressure. No major changes in c, T' or λ were noted. Stimulation of the n. depressor cordis had an extremely depressing effect on the blood /136 pressure, but there was not much change in c, T' or λ.

Tension on the lower limb increased both P_s and P_d, but no change in the tendency of c, T' and λ to hold constant was noted. Asphyxia in general increased the blood pressure, but no change in the constant values of c, T' or λ was noted. Bloodletting decreased the blood pressure while c decreased, T' increased, and λ had a tendency to decrease. For the other factors, this type of treatment either modified the tendencies to constant values or caused the appearance of changes of indeterminate kind. Since it is not the object of this paper to report on them, we shall confine ourselves to giving the actual values for two typical examples in Table 1.

The relationship between these several quantities occupies the focal /142 point of interest of this study; in order to contribute to the investigation of the relationships postulated in the past on the basis of critical discussion of theories on the fundamental vibration, and of dynamic models as well, we selected the following factors and subjected the correlations between them to mathematical investigation: ($\lambda \sim c$, $\lambda \sim cS$, $\lambda \sim W$, $\lambda \sim \frac{W}{c}$, $W \sim \frac{P_2}{P_1}$, $\frac{P_s - P_d}{P'_s - P'_d} \sim$ $\sim \frac{P_2}{P_1}$). We also calculated the regression lines. Figures 2a to Fig.3f give

examples of each measured value by means of a graph, while Table 2 gives the calculated values. All that has been done here is to investigate whether or not the relationship between two quantities, expressed either by a correlation coefficient or by a regression line, is linear. Thus, no accurate judgment can be formulated on this basis, although we do believe that there can be no question but that consideration within the framework of the method of least squares will provide a clue.

TABLE 1a

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THE VARIOUS FACTORS OF THE ARTERIAL PULSE OF RABBITS

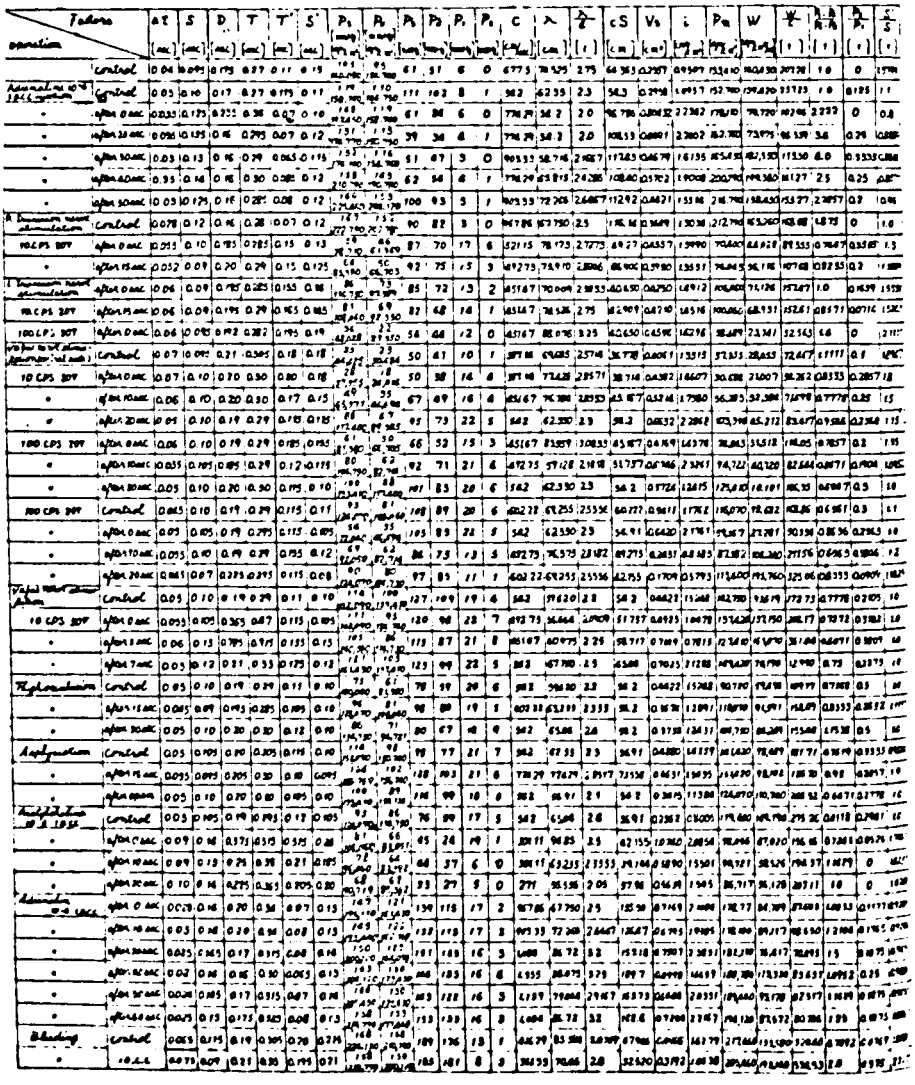

TABLE 1b

THE VARIOUS FACTORS OF THE ARTERIAL PULSE OF DOGS

Factors / operation	at	S	D	T	T'	S'	P_a	P_d	P_1	P_2	P_3	P_4	C	λ	$\frac{S}{T}$	$\frac{cS}{T}$	V_0	t	P_m	W	$\frac{P_m}{W}$	$\frac{t}{t_0}$	$\frac{P_m}{P_a}$	$\frac{T}{T'}$
		sec	sec	sec	sec	sec	mm Hg	mm Hg	mm Hg	mm Hg	mm Hg	mm Hg	cm/sec	cm	(%)	(cm)	cm³	cm	mm Hg		(%)	(%)	(%)	(%)
Ligation of the ENT system	Control	0.065	0.11	0.19	0.50	0.18	0.15	116	111	173	159	91	9	595 535	166.8	41.0	65 3007	0.0871	36.293	282.91	747.306	12.849	1.36048	0.3905	0.38
	after 0 sec	0.025	0.125	0.225	0.35	0.125	0.135	210	187	273	199	58	3	1968	81.66	5.0	0.33	25.7134	44.295	218.83	1.487.19	6.636	1.3948	0.6030	0.40
"	after 10 sec	0.04	0.12	0.235	0.355	0.135	0.16	211	182	281	192	62	0	66.75	0.34175	3.125	80.1	17.864	48.643	291.251	1.499.75	9.382	1.4087	0	0.4087
"	after 20 sec	0.055	0.115	0.265	0.36	0.125	0.085	221	186	262	180	62	0	162 857	118.2679	4.286	97.178	17.2976	51.1683	262.91	1.357.79	10.956	1.4838	0	1.36.49
Constant artery clamp	Control	0.055	0.11	0.175	0.285	0.165	0.16	169	164	173	166	28	6	182.857	125.714	6.1743	83.948	23.599	51.487	428.794	1.427.69	7.115	1.5817	0.3463	1.4948
"	after 0 sec	0.06	0.11	0.175	0.285	0.175	0.17	196	161	198	152	26	8	66.75	160.125	6.775	73.425	41.196	35.506	178.93	1.498.88	7.515	1.7367	0.3977	1.5483
"	after 15 sec	0.055	0.095	0.175	0.27	0.15	0.145	220	160	230	177	32	7	162.857	116.4286	6.2857	72.471	9.7534	35.869	253.78	1.401.33	9.217	1.8182	0.2499	1.5189
"	after 30 sec	0.055	0.11	0.225	0.335	0.16	0.15	215	171	221	173	39	7	162.857	122.857	6.5714	83.945	11.483	39.178	265.78	1.401.30	9.382	1.7917	0.1796	1.3838
Vagus nerve cut	Control	0.06	0.10	0.165	0.245	0.16	0.155	229	211	219	211	29	7	66.75	146.8	6.0	66.75	38.1782	38.178	238.63	1.446.91	11.0999	1.6429	0.2598	1.66
"	after 0 sec	0.065	0.11	0.26	0.53	0.16	0.16	225	196	236	191	37	8	195.393	141.9383	3.5536	65.286	42.595	27.1848	215.492	1.440.28	15.00	1.6965	0.2182	1.6549
"	after 10 sec	0.062	0.105	0.18	0.285	0.18	0.17	233	181	205	166	61	8	165.798	116.428	4.2857	66.75	5.1987	38.0238	28.489	833.71	1.3907	1.1383	0.1995	1.6149
"	after 20 sec	0.05	0.11	0.20	0.31	0.21	0.16	236	196	248	171	33	7	588	118.06	4.1210	57.66	38.4296	33.6863	287.609	1.545.38	11.8975	1.6468	0.2127	1.6965
Vagus and Sympathetic (control value)	Control	0.065	0.11	0.19	0.30	0.17	0.16	217	184	261	177	21	9	595 533	165.882	3.7778	62.365	36.648	25.6863	215.38	1.427.7	17.321	1.3333	0.4738	1.7727
10 CPS 20V	after 0 sec	0.065	0.105	0.20	0.305	0.165	0.15	227	195	198	169	26	10	595 535	169	5466.7	62.3	7.2435	23.1861	287.698	1.282.27	12.396	1.1934	0.3846	1.6398
"	after 10 sec	0.05	0.105	0.195	0.30	0.17	0.155	215	181	192	162	27	9	536	99.08	3.6	55.62	3.8276	31.6459	215.535	1.406.25	15.0	1.3193	0.4781	1.6161
"	after 20 sec (Sympathetic)	0.05	0.11	0.185	0.295	0.16	0.16	218	189	193	166	27	11	536	103.86	3.2	57.66	7.9719	32.0197	261.129	1.397.61	11.5281	1.4974	0.4117	1.6496
50 CPS 20V	after 0 sec	0.065	0.11	0.19	0.30	0.175	0.17	196	152	180	146	31	8	595 533	133.833	3.9999	65.367	7.9185	36.0617	238.129	1.367.61	10.5281	1.4974	0.4781	1.7838
"	after 10 sec	0.065	0.105	0.185	0.29	0.18	0.18	218	167	168	140	29	6	595 535	146.8	4.0	62.3	7.4948	25.4798	217.498	1.401.96	10.3535	1.6	0.3548	1.6083
"	after 20 sec (Sympathetic)	0.064	0.11	0.185	0.295	0.16	0.16	196	160	193	156	25	8	662.857	91.8999	5.6164	66.75	3.2370	27.857	228.196	1.448.12	10.1995	1.2383	0.332	1.6185
100 CPS 20V	after 0 sec	0.065	0.105	0.20	0.305	0.185	0.18	196	152	165	132	29	9	595 535	107.667	4.1111	62.3	7.972	28.395	235.795	1.441.88	10.2645	1.4645	0.3108	1.6933
"	after 10 sec	0.06	0.104	0.176	0.28	0.16	0.165	209	163	168	145	25	8	667.5	116.8	4.0	59.62	5.87	33.864	236.119	1.432.67	10.7906	1.3649	0.3633	1.6875
"	after 20 sec (Sympathetic)	0.05	0.10	0.196	0.295	0.175	0.15	217	174	185	168	22	9	536	91.7	3.5	53.6	3.2706	27.0577	248.169	1.416.75	11.049	1.6	0.4691	1.8
Sympathetic Excitation (Constant value)	Control	0.05	0.10	0.19	0.30	0.175	0.15	181	163	205	176	29	9	536	91.7	3.5	53.6	3.9288	29.8277	236.126	1.251.38	8.4534	1.5703	0.4693	1.8
30 CPS 20V	after 0 sec	0.05	0.11	0.19	0.30	0.175	0.156	208	168	180	148	30	10	536	91.7	3.5	53.6	3.9288	29.8277	236.126	1.251.38	8.4534	1.5703	0.4693	1.8
"	after 10 sec	0.065	0.10	0.19	0.30	0.165	0.165	206	174	180	148	29	9	595 535	99.9	5466.7	59.595	8.045	29.78	236.78	1.440.62	10.1746	1.235	0.3409	1.68
"	after 20 sec	0.065	0.10	0.19	0.305	0.175	0.15	205	166	186	157	30	11	595 535	103.833	3.8889	59.595	8.2630	27.498	215.195	1.396.72	8.3636	1.5373	0.3373	1.6
"	after 30 sec	0.066	0.105	0.18	0.285	0.155	0.155	185	163	185	156	27	9	660.818	98.4548	3.5227	63.729	9.046	32.6	218.793	1.419.42	11.0909	1.3182	0.3535	1.6818
Right occlusion	Control	0.05	0.10	0.19	0.29	0.18	0.16	189	166	176	143	33	11	536	96.32	3.6	52.4	4.0776	31.1128	249.999	1.396.7	10.2857	1.3836	0.3983	1.6
"	after 0 sec	0.05	0.105	0.19	0.295	0.196	0.166	196	157	179	150	29	9	536	101.436	3.88	55.02	11.478	30.196	206.305	1.398.18	9.8571	1.5515	0.3973	1.7903
"	after 15 sec	0.065	0.105	0.175	0.28	0.155	0.15	206	152	165	118	25	8	595.893	91.8647	3.6666	62.3	12.3686	44.7256	271.879	1.378.24	8.5796	1.9259	0.32	1.6308
"	after 30 sec	0.05	0.095	0.19	0.285	0.175	0.155	196	91	125	99	35	11	536	91.7	3.5	49.98	11.2817	41.548	454.86	1.429.81	6.4444	1.5343	0.3963	1.6316
Left occlusion	Control	0.065	0.10	0.185	0.285	0.18	0.165	199	153	181	137	33	11	595 535	116.8	4.8	57.993	10.665	33.995	228.799	1.395.25	10.368	1.3956	0.3898	1.667
"	after 15 sec	0.05	0.09	0.195	0.285	0.17	0.155	191	97	151	118	33	11	536	99.08	3.6	47.16	5.4975	29.996	183.685	1.401.69	9.3888	1.3121	0.5395	1.7228
"	after 30 sec	0.06	0.105	0.19	0.295	0.155	0.16	197	177	181	148	30	10	667.5	109.462	3.875	72.907	7.5426	42.2361	298.619	1.395.69	14.726	1.4739	0.3945	1.8398
Left occlusion of 4 arteries	Control	0.064	0.11	0.19	0.30	0.18	0.15	218	175	211	178	31	9	595 533	164.29	3.4286	72.4768	10.792	36.3278	295.989	1.448.81	8.799	1.2387	0.4089	1.6488
"	after 0 sec	0.06	0.09	0.12	0.21	0.15	0.125	215	215	207	227	70	9	667.5	98.775	5.29	80.875	14.9798	71.9428	375.58	1.478.57	12.8955	1.4897	0.3582	1.6889
"	after 10 sec	0.06	0.096	0.18	0.276	0.165	0.16	203	219	66	10	667.5	161.175	3.575	62.365	35.808	9.5746	357.609	1.4572	2.4548	2.8588	0.8619	1.4896		
"	after 20 sec	0.06	0.095	0.125	0.22	0.165	0.15	216	211	62	9	655.5	96.7875	5.625	61.8675	13.5648	5.3625	345.129	1.4589	1.1167	0.4882	1.6789			
"	after 30 sec	0.06	0.10	0.16	0.26	0.155	0.16	205	195	53	7	667.5	113.625	3.875	66.75	11.869	7.5648	388.539	1.4738	0.2368	1.5	0.1521	1.6		
Left occlusion of ALL system	Control	0.053	0.115	0.19	0.345	0.195	0.18	212	170	189	168	19	7	888.666	98.864	2.5	58.27	11.2218	35.7987	262.457	1.731.19	8.4598	1.6667	0.4688	1.6462
"	after 20 sec	0.06	0.065	0.21	0.275	0.21	0.105	184	98	134	92	12	2	665	93.85	1.8955	38.925	6.3097	18.6478	141.495	1.484.4	10.959	1.2779	0.3667	1.6188
Bleeding	Control	0.05	0.11	0.19	0.31	0.225	0.195	172	118	160	119	26	5	536	177.7	4.5	3.566	46.497	28.1488	88.196	1.348.68	9.8916	1.9908	0.3818	1.7097
"	40 cc	0.06	0.09	0.19	0.28	0.19	0.095	144	85	105	90	9	0	665	85.55	8.1898	66.69	7.5222	25.5097	178.342	1.498.15	11.2872	1.7583	0	1.6838

t.....The time difference between the beginning of the aorta pulse and the femoral pulse.
S.....Duration of the systole.
D.....Duration of the diastole.
T.....Pulse time.
T'.....Period of the fundamental vibration.
S'.....Duration between the beginning and incisura of the femoral pulse.
Pa.....Systolic pressure of the aorta.
Pd.....Diastolic pressure of the aorta.
P'a.....Systolic pressure of the femoral artery.
P'd.....Diastolic pressure of the femoral artery.
P_1 & P_2.....Pressure as shown in Fig. 1, a and Fig. 1, b.
C.....Pulse wave velocity.
λ.....Wave length of the fundamental vibration.
l.....Pulse conduction length.
Vo.....Cardiac stroke volume per beat.
t.....Mean ejection velocity.
Pm.....Mean aortic pressure.
W.....Total effective peripheral resistance.

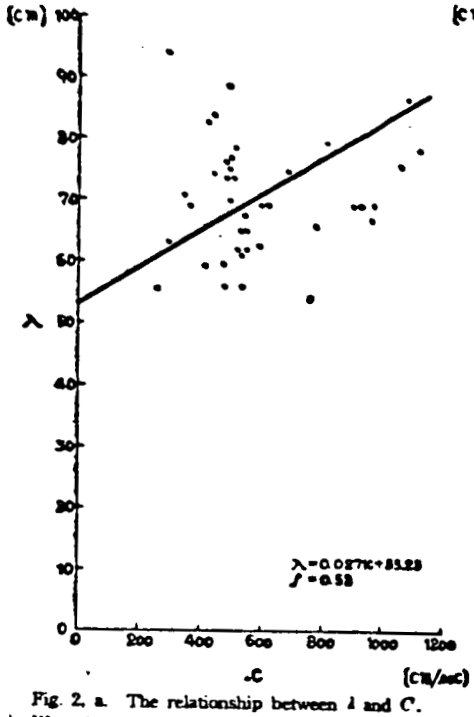

Fig. 2, a. The relationship between λ and C.
λ···Wave length of the fundamental vibration.
c···Pulse wave velocity.
ρ···Correlation coefficient.

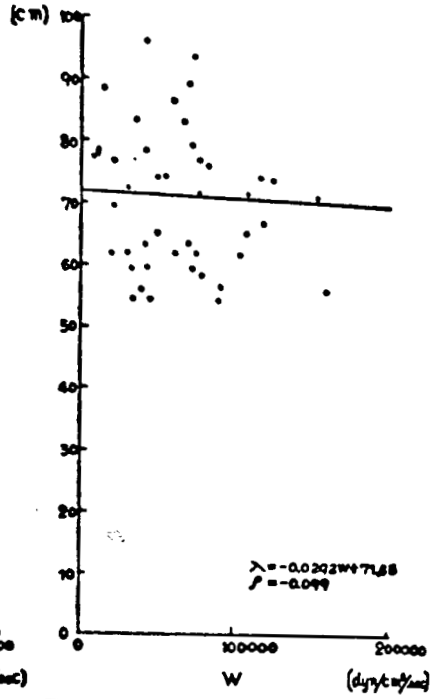

Fig. 2, c. The relationship between λ and W.
λ···Wave length of the fundamental vibration.
W···Total effective peripheral resistance.
ρ···Correlation coefficient.

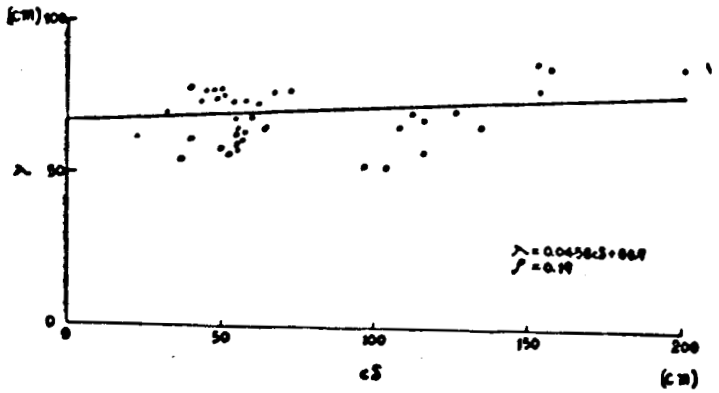

Fig. 2. b. The relationship between λ and cS.
λ···Wave length of the fundamental vibration.
c···Pulse wave velocity.
S···Duration of the systole.
ρ···Correlation coefficient.

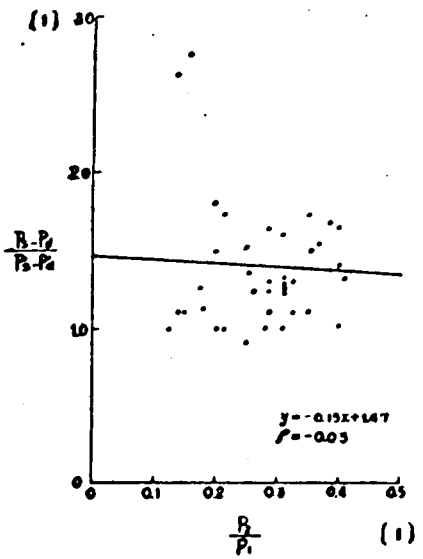

Fig. 2, f.

The relationship between $\frac{P_s - P'd}{P's - P'd}$ and $\frac{P_2}{P'_1}$

P_1 & P_2'···Pressure as shown in Fig. 1, a and Fig. 1. b.

Ps···Systolic pressure of the aorta.

$P's$···Systolic pressure of the femoral artery.

Pd···Diastolic pressure of the aorta.

$P'd$···Diastolic pressure of the femoral artery.

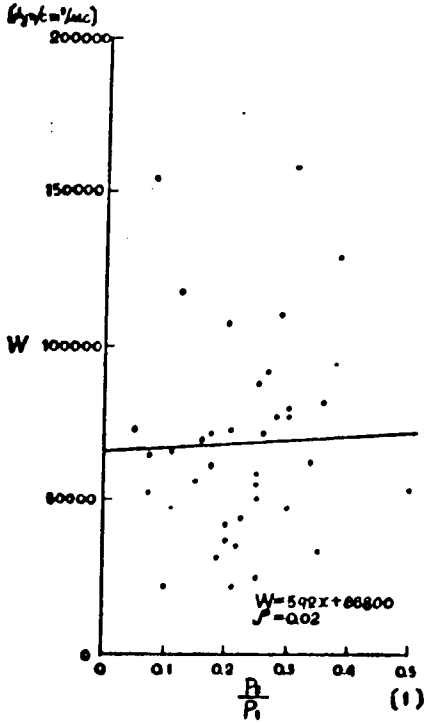

Fig. 2, e.

The relationship between W and $\frac{P_2}{P'_1}$

W···Total effective peripheral resistance.

P'_1 & P'_2···Pressure as shown in Fig. 1 a and Fig. 1, b.

ρ···Correlation coefficient.

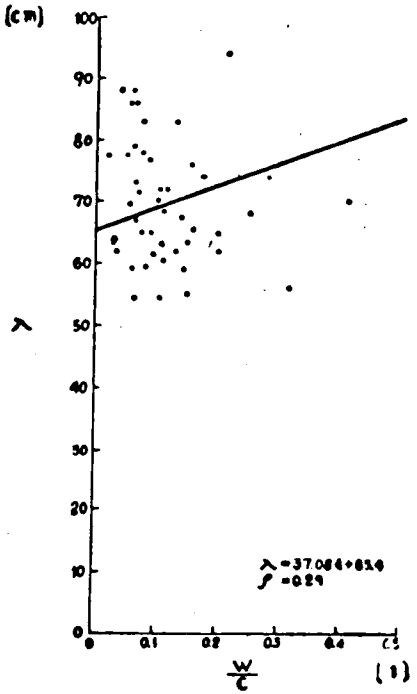

Fig. 2, d.

The relationship between λ and $\frac{W}{c}$

λ···Wave length of the fundamental vibration.

W···Total effective peripheral resistance.

c···Pulse wave velocity.

ρ···Correlation coefficient.

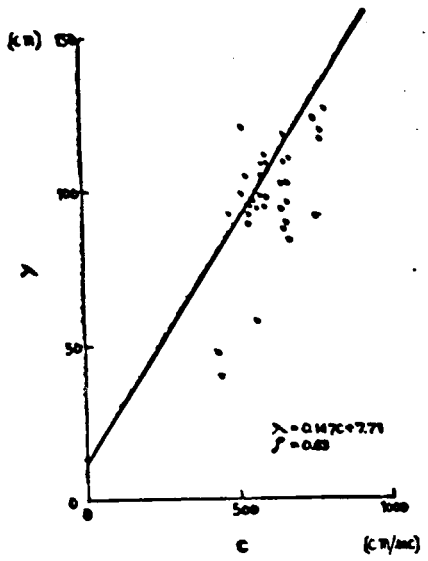

Fig. 3, a.
The relationship between λ and c.
λ···Wave langth of the fundamental vibration.
c···Pulse wave velocity.
ρ···Correlation coefficient.

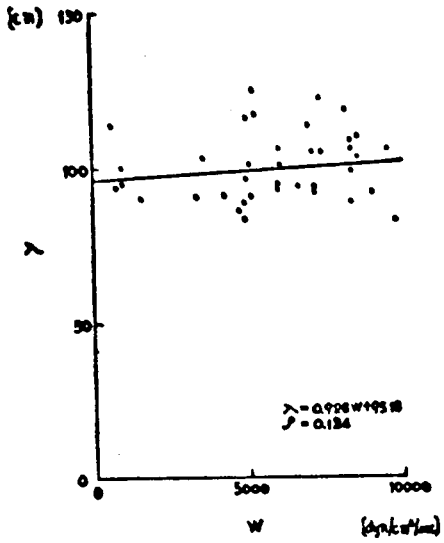

Fig. 3, c. The relationship between λ and W'.
λ···Wave length of the fundamental vibration.
W'···Total effective peripheral resistance.
ρ···Correlation coefficient.

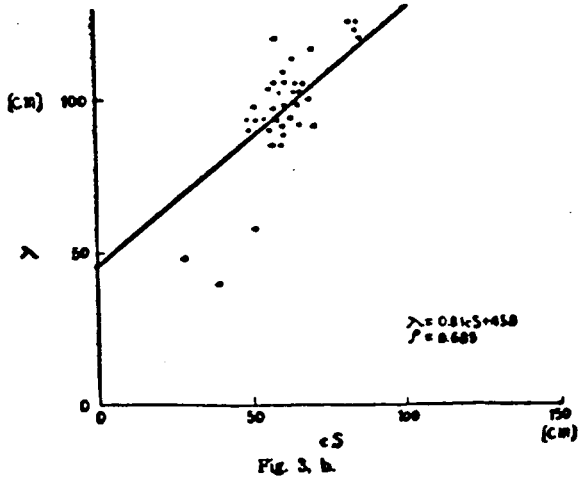

Fig. 3, b.
The relstionship between λ and cS.
λ···Wave length of the fundamental vibration.
c···Pulse wave velocity.
S···Duration of the systole.
ρ···Correlation coefficient.

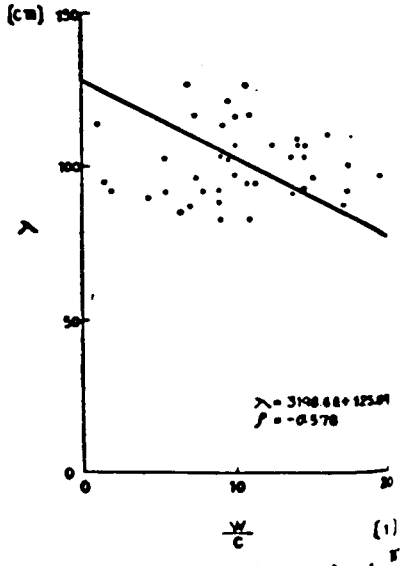

Fig. 3, d. The relationship between λ and $\frac{W'}{c}$.
λ···Wave length of the fundamental vibration.
W'···Total effective peripheral resistance.
c···Pulse wave velocity.
ρ···Correlation coefficient.

Table 2.

Correlation coefficient and regression line of various factors.

		Correlation coefficient	Regression line
(1) $\lambda \sim c$	Dog	① +0.79	$\lambda = 0.1996c + 216$
		② +0.63	$\lambda = 0.167c + 776$
		③ +0.437	$\lambda = 0.0448c + 9587$
	Rabbit	① +0.285	$\lambda = 0.02c + 566$
		② +0.72	$\lambda = 0.002c + 303$
		③ +0.52	$\lambda = 0.0771c + 5375$
(2) $\lambda \sim w$	Dog	① +0.73	$\lambda = 3384w + 8576$
		② +0.124	$\lambda = 0.926w + 9518$
		③ −0.109	$\lambda = -0.767w + 1319$
	Rabbit	① +0.027	$\lambda = 0.0199w + 667$
		② +0.21	$\lambda = 0.11w + 6245$
		③ −0.099	$\lambda = -0.0792w + 7183$
(3) $\lambda \sim \frac{W}{c}$ $(\frac{W}{c} \sim \lambda)$	Dog	① +0.18	$\lambda = 5762\lambda + 9686$
		② −0.576	$\lambda = -3192\lambda + 12549$
		③ +0.155	$\lambda = 1178\lambda + 11919$
	Rabbit	① −0.07	$\lambda = -1611\lambda + 690$
		② −0.14	$\lambda = -516\lambda + 7739$
		③ +0.29	$\lambda = 3708\lambda + 654$
(4) $\lambda \sim cS$	Dog	① −0.17	$\lambda = -0.156cS + 1204$
		② +0.685	$\lambda = 0.18cS + 458$
		③ −0.607	$\lambda = -0.07cS + 1769$
	Rabbit	① +0.52	$\lambda = 0.212cS + 5209$
		② +0.718	$\lambda = 0.30cS + 313$
		③ +0.19	$\lambda = 0.0458cS + 669$
(5) $w \sim \frac{P_2}{P_1}$ $(\frac{P_2}{P_1} \sim x)$	Dog	① +0.167	$w = 4837x + 6780$
		② +0.349	$w = 7446x + 2910$
		③ +0.19	$w = 44x + 3250$
	Rabbit	① +0.42	$w = 14234x + 35200$
		② +0.325	$w = 1253x + 30300$
		③ +0.02	$w = 592x + 66000$
(6) $\frac{P_1-P_2}{P_1-P_2} \sim \frac{P_2}{P_1}$ $\left(\frac{P_1-P_2}{P_1-P_2} \sim y,\ \frac{P_2}{P_1} \sim x\right)$	Dog	① +0.16	$y = 0.739x + 145$
		② −0.03	$y = -0.15x + 167$
		③ −0.398	$y = -2.79x + 142$
	Rabbit	① −0.19	$y = -0.0926x + 1036$
		② +0.556	$y = 3.883x - 0.395$
		③ −0.067	$y = -0.099x + 0.916$

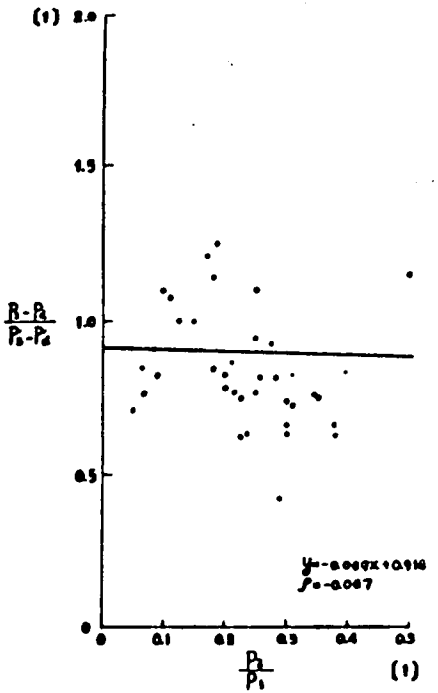

Fig. 3, f.

The relationship between $\frac{P_s - Pd}{P'_s - P'd}$ and $\frac{P_2}{P_1}$

P_1 & P_2···Pressure as shown in Fig. 1, a and Fig. 1, b.

P_s···Systolic pressure of the aorta.

P'_s···Systolic pressure of the femoral artery.

Pd···Diastolic pressure of the aorta.

$P'd$···Diastolic pressure of the femoral artery.

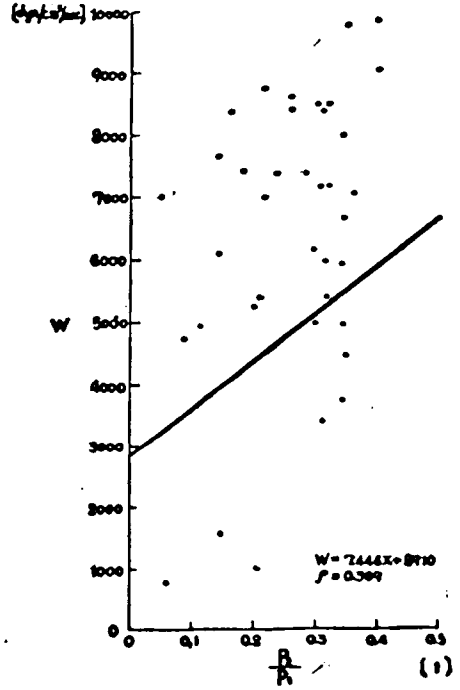

Fig. 3, e.

The relationship between W and $\frac{P_2}{P_1}$

W···Total effective peripheral resistance.

P_1 & P_2···Pressure as shown in Fig. 1, a and Fig. 1, b.

ρ···Correlation coefficient.

III. DISCUSSION

The most logical thesis in the dynamic theory of the fundamental vibration is to be found in the vibration theory first advanced by Frank, which is based on the existence of a standing wave, as more particularly demonstrated by Hamilton and Dow (Bibl.10), and which appears to become more and more entrenched. Indeed, by following this theory in practice, and taking 1/2 λ [Frank (Bibl.8)] or 1/4 λ [Wezler and Böger (Bibl.23)] as the effective length of the air chamber of a blood vessel, a dynamical application of the circulation rate is now in practice supplied. Broemser (Bibl.5) derived the period of this vibration from the dynamical theory and argues in favor of its correlation with the peripheral resistance. According to him, this period becomes shorter with increasing peripheral resistance. However, the dynamical model taken by Broemser (Bibl.4) as his theoretical foundation is certainly an object of the vibration theory - something in the nature of a concentration network, so to say. It is not an object of wave-motion theory, nor something in the theory of distribution-constant networks. In this connection, Hatakeyama (Bibl.11), in applying the theory of vibrations in an elastic tube to the pulse wave, reports that the wavelength may vary within a range of a factor of 2 with changes in the peripheral resistance. According to the conventional dynamical theory, the wavelength of a vibration in a closed tube, i.e., in a tube closed at both ends, is twice the length of the tube, and in an open tube, i.e., in a tube closed at one end and open at the other end, the wavelength of a vibration is four times the tube length. Frank (Bibl.8) adopted the former proposition, while Wezler and Böger accepted the latter. However, as will be easily understood from the example of a wind instrument, a tube, even though it may be called a closed tube, will at some point not stop the flow of air; since the impedance will

vary with the size of the opening and the length of the tube, the wavelength, though it may not be twice the tube length, may also take a value other than four times that length (Bibl.13). Hatakeyama emphasizes this point and shows that, in a vascular system closely resembling a closed tube (for instance, the system of major arteries in the upper extremities), the wavelength increases with the resistance and that Broemser's theory, postulating that the wavelength decreases with increasing resistance, is not necessarily true (Bibl.11). Wezler et al. (Bibl.21, 23), based on the simple consideration that an increase in peripheral vascular resistance, because of contraction of the blood vessels, would decrease the size of the thick portions of the blood vessels, still believe that an increase in resistance would decrease the wavelength, in disagreement with Hatakeyama's position.

Hatakeyama experimentally investigated this point on several examples. He stated that there are various cases of vibration in the aorta and that, depending on the animal, there are marked individual differences and variations by factors that at most can reach 2. To be sure, Wetterer and Deppe (Bibl.17, 18) give figures in their research reports showing that λ may vary by a factor of 2 in the very same individual. Recently, however, Kapal, Martini and Wetterer (Bibl.14) and Bleichert, Lezgus and Martini (Bibl.3), in discussing the relationship of λ and the blood-vessel length for the fundamental vibration of the pulse wave in the human femoral and radial arteries, investigated the position of the loops and nodes of the vibration and reported no extreme variation.

Now, according to the experimental results of the present author, λ may undoubtedly vary considerably, even in the same individual. For example, in the rabbit, the ratio of the maximum to minimum λ in the same individual was 2.60, 2.96, 1.62, etc., and in the dog it was 2.02, 3.14, 1.89 and so on. This is a

range of variation for λ by a factor of more than 2. The calculated values of λ, however, were here obtained by multiplying the period of the fundamental vibration T' by the pulse wave transmission velocity c, measured by a certain fixed method. Strictly speaking, since the transmission rate might vary with the location of the blood vessel, the position of the wave in the segment of the blood vessel cannot be exactly specified from the value of this λ. However, since the factors, for the most part, are no doubt entirely suited to their purpose, a comparison of this λ with the anatomic length of the aorta L indicates that on the whole it was valid within the range from 2L to 4L, not being very much shorter than 2L nor very much longer than 4L. From the viewpoint of wave-motion theory, this statement is almost free of contradiction. A /143 glance at the measured and calculated values indicates that the transmission velocity is extremely variable. In some cases, the ratio of its maximum to its minimum is as high as 6, which is greater than the corresponding ratio for λ. However, if the period of the fundamental vibration is also governed by factors other than the intravascular vibration, for the above causes, then it would be possible for λ to vary substantially with c. In fact, a study of the correlation between λ and c does show a positive correlation of 0.79 according to the experimental results. In some cases, however, it is below 0.3, so that no unqualified conclusion can be drawn, and, for instance from the wave-motion theory, the correlation between the elastic elements of the blood vessels cannot be neglected in the determination of wavelength.

As stated above, according to the wave-motion theory there should be a close relationship between peripheral resistance and wavelength. However, it is difficult to express the peripheral resistance, as such, quantitatively and accurately. Moreover, a kind of ejection-point impedance would be obtained by

an accurate measurement of the so-called effective peripheral resistance (mean blood pressure versus mean flow velocity), and since nothing is known on the distribution of the resistance at each position of the blood vessel, the situation grows more and more complicated. Consequently, there are many questionable points relative to the values of W that I have calculated. For example, W has the meaning of overall effective peripheral resistance, and in order to calculate it accurately it is necessary to measure accurately the mean flow velocity, and consequently to measure accurately the cardiac output. However, it is extremely difficult to make accurate measurements of the cardiac output, especially the cardiac output for a single stroke. Wetterer and Deppe (Bibl.18) made continuous recordings of the circulation flow curve or velocity curve in the primitive aorta, using a magnetic flowmeter, thus directly measuring the cardiac stroke volume. But even by using such a high-confidence method, these authors were unable to measure the blood flow entering the system of coronary blood vessels and wound up with negative loads, confusing the thoracotomy and the other dynamic conditions. On the other hand, dynamic methods of measuring the cardiac output, such as those used here, do have the advantage over chemical methods that it is possible, from time to time and from instant to instant, to measure the cardiac output for a single stroke, although the confidence level may not be entirely satisfactory. But there were no other reliable methods that one could use instead, and therefore I used these. There are many dynamic methods besides those of Broemser-Ranke (Bibl.4) that were here employed: Wezler and Böger (Bibl.23), Frank (Bibl.8), Bazett et al. (Bibl.2), Hamilton et al. (Bibl.10, 16), Wetterer (Bibl.19), Recklinghausen (Bibl.15), and Hatakeyama et al. (Bibl.12). While their confidence level is high, the operations are complicated, and the amount of calculation required would have been entirely

out of the question for this project. We believe that comparatively accurate measurements can be obtained by the Broemser-Ranke method, considered both theoretically [Hatakeyama (Bibl.12)] and experimentally [Wetterer and Deppe (Bibl.16)].

Now, on investigating the relationship between λ and W, we find cases of positive correlation and others of negative correlation. In either case they may run up to +0.21 with about ±0.1. The correlation between these two quantities must therefore be termed unsubstantial. The causes are unknown. As already stated, the value of W is unstable. Since the arterial system of the aorta exhibits considerable vibration at the branch points, it does not seem reasonable that there should be no close correlation observable. Since, further, the relationship between λ and W in a theoretical model is complex, it cannot be definitely asserted that there is no interrelation between them, even though the linear correlation may be tenuous. Of course, it is not entirely clear whether the theoretical results shown by a second-order partial differential equation would not, by comparison, be preferable, but they are extremely complex and since I felt them to be rather remote from practical problems I did not examine them in detail.

Next in the theory of λ varying with W, let some factor other than the peripheral resistance be constant; for example, let c, etc., be constant. If c varies, as will be clear from the Hatakeyama theoretical formula, then, since W/c must be considered to be one variable, it would be more reasonable to investigate the relation between λ and W/c. Thus when we investigate the correlation between λ and W/c, we find that while the corresponding correlation coefficient is -0.576, we still cannot say, on the whole, that this correlation is particularly high. Even in this case, however, since the relation between λ

and W is not thought to be linear, the existence of a close correlation between the two quantities cannot be denied on the basis of these facts alone.

The effective length of the air chamber was first given as cS by Broemser, but Aub (Bibl.1) later investigated this subject experimentally, and established it at 0.5 cS for many animals. On the other hand, Wezler and Böger took the /144 effective length as $\lambda/4$, in contrast to Frank's earlier finding that it was $\lambda/2$. Thus, there is a great difference between the method of determining the cardiac output given by Broemser and Ranke and that used by Wezler and Böger, which implies the possibility of showing that the two methods yield very different values, provided there is no close correlation between cS and λ.

In view of this situation, an examination of the correlation coefficients found by us between λ and cS shows positive correlation in some cases, and negative correlation in others, with a coefficient of correlation above 0.5 in only a few cases. However, the fact that there are cases with a coefficient of correlation below 0.2 as well as cases of negative correlation means that entirely separate figures should be used for treating cS and λ.

In general, in any discussion of vibration, not only the period, wavelength and amplitude are involved but also the degree of damping. If the fundamental vibration is taken as a type of hypervibration, it should be possible to express the degree of its damping by the ratio between the first and second peaks. Strictly speaking, of course, if we are concerned with the damping as an index number, ordinary common sense would tell us to use the so-called logarithmic damping decrement, but here, purely for convenience, we have adopted the ratio P_2/P_1 as our measure of the damping conditions. The first factor to be considered in connection with the damping is the peripheral resistance. If we do not consider what the relationship with the loops and nodes of the standing-wave

vibration of the region of the femoral artery might be, then it is reasonable to suppose, as Broemser has shown, that as the peripheral resistance increases the damping will decrease and P_2/P_1 will approach 1. The fact that, in reality, all the correlation coefficients between W and P_2/P_1 are positive is not inconsistent with this idea. However, if these values do not become excessively great, it is difficult to expect these correlation coefficients to be accurate. Considered within the framework of the wave-motion theory, the fact that P_2/P_1 increases with W is connected with the fact that, as W increases, the measured portion of the femoral artery would approach the vibration loop, so that there should be a negative correlation between λ and W. Nevertheless, as we stated at the beginning of this discussion, these cases are not necessarily confined to negative correlations, and thus this point need not be overemphasized.

Passing now to the correlation between the ratio of the amplitudes of the pressure vibrations in the aorta and femoral artery and the ratio P_2/P_1, the same point is noted. Some correlation coefficients are positive, others negative, and in some cases the absolute values of the correlation coefficients are less than 0.1. Nevertheless, one cannot say per se that an increase in amplitude of the vibration region of the femoral artery is parallel to a marked fundamental vibration.

The view that it is the ejection of blood from the heart that promotes the fundamental vibration has been advanced and discussed above. Another explanation given in the past is that the second peak or dicrotic rise is a reaction to the closing of the semilunar valves [Wiggers et al. (Bibl.24)]. If this is so, then the period S' between the trough of the fundamental vibration of the femoral artery, i.e., the incisure, and the initial part of the pulse wave would have to be almost identical with S. Thus, S'/S would have to be practi-

cally equal to 1. The values actually measured, however, range from 0.7 to 2 in both rabbit and dog, and in some cases even reach 4, so that S' can definitely not be considered as being substantially the same as S, in value.

Wehn (Bibl.20) holds that the vibration amplitude of the peripheral arterial blood pressure is at least twice that of the central arterial blood pressure, and that the reaction of the organism to this and other causes might give rise to the vibration of blood pressure; but our own measurements show no examples of such phenomena. If we consider the accuracy of measurement and the complexity of the vascular system, we cannot here argue for the agreement or disagreement of the refined figures, and it must, in particular, be considered that the blood is ejected from the heart during the systolic period and that, consequently, there must be a close relationship between the form of the blood-pressure wave during this period and the properties of the myocardium. For the fundamental vibration period to be a proper object of vibration theory study by the methods used in the present work, the ejection of the blood would have to obey a square law. Unless it does, the summit of the first peak due to the ejection pattern of the blood during the systole must vary. This was the analysis given long ago by Frank (Bibl.8), and later by Wetterer (Bibl.19), to the effect that the first peak due to the blood ejection pattern might occur either earlier or later than the ideal pattern. Later, with respect to the second peak, the blood is continuously ejected in the same way, obeying a square law. In comparison with this pattern, after the closing of the aortal valves, the blood ejection pattern drops to zero, so that it cannot be considered to be simply the second wave of the after-vibration. It can easily be shown, even in model experiments, that in general the second peak has an actual vibration which leads that anticipated from the ideal vibration pattern. Consequently, the theory

mentioned at the very beginning will be supported as compared with the view that, on variation of L within the interval of 2 to 4 times, C varies rela- /145
tively little. In essence, the former theory states that the fundamental vibration is excited by the ejection of blood from the heart. However, λ, as considered by many investigators up to now, is not a fixed quantity but may vary by a factor of 2 or more, and the dynamic model must be considered to be sometimes that of an open tube, sometimes that of a closed tube, and sometimes intermediate between these. In the case of animal experiments, however, very great changes in the condition of the circulatory system can be obtained by the application of very great loads to it; in experiments on human subjects, such as those performed by Kapal, Martini and Wetterer (Bibl.14) and by Bleichert, Lezgus and Martini (Bibl.3), since it was impossible to produce extreme changes, the relationships found for λ were practically constant.

The blood vessels of the arm, considered as an open tube opening into the wide cavity represented by the aorta, would likewise be expected not to show extreme changes in λ, since the distribution of peripheral resistance is not too complex. Yet Wezler et al. (Bibl.21) have shown that cooling of the hand and similar treatment induces substantial changes in the λ of the pulse wave of the brachial artery, and Wezler et al. (Bibl.22) have also found that the ratio of the λ of the pulse wave of the brachial artery to the length of the blood vessel decreases with increasing age of the subject. The latter fact cannot be explained by the change in the actual length of the blood vessels which many researchers believe to occur, since actually, with increasing diameter of the blood vessel and thus with decreasing peripheral resistance, λ is said to decrease; this can be explained instead by an approach from the closed tube system to the open tube system.

IV. CONCLUSIONS

To investigate the causes of the fundamental vibration of the arterial pulse wave, the correlations between the following pairs of elements of the blood-pressure curves for the aorta and femoral artery of the dog and rabbit were determined:

$$1)\ \lambda \sim c;\quad 2)\ \lambda \sim cS;\quad 3)\ \lambda \sim W;\quad 4)\ \lambda \sim \frac{W}{c};$$

$$5)\ W \sim \frac{P_2}{P_1};\quad 6)\ \frac{P_s - P_d}{P_s' - P_s'} \sim \frac{P_2}{P_1}$$

where

c = transmission velocity of the aortal wave;

λ = wavelength of the fundamental vibration;

S = duration of the aortic systole;

W = effective peripheral resistance;

P_s and P_s' = the systolic pressures in the aorta and femoral artery, respectively;

P_d and P_d' = the diastolic pressures in the aorta and femoral arteries, respectively;

P_2 and P_1 are given in Fig.1 (which see).

As a result we have learned that the fundamental vibration is caused primarily by the vibration accompanying the ejection of blood from the heart into the arterial system, and that its wavelength varies greatly with the pattern of such ejection and with the dynamical state of the vascular system. Various theories heretofore advanced which give the effective length of the air chamber as the basic cause of this phenomenon are critically examined.

We express our profound gratitude to Professor Ippei Hatakeyama for his

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